cases which cannot always be differentiated by ordinary clinical means.

It must also be remembered that in infancy and early childhood the classical signs may be absent or incomplete and a definite diagnosis must in some instances be postponed until the child grows older, or the procedures outlined previously are utilized.

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QUESTION AND ANSWER

Question: In the absence of any physical findings in infants and children, what are the indications for diodrast studies or venous catheterization?

DR. ROBINSON: In the absence of any physical signs or symptoms, I do not think you could even suspect heart disease—there would be no indication for diodrast.

Metatarsus Varus

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METATARSUS varus, a common finding in infants, may be of very slight degree or it may be so marked as to make one consider pes equinovarus in the differential diagnosis. The condition is usually neglected unless the deformity is very striking, as there is a tendency for the foot to straighten, thus giving a more normal external appearance. Apparently the foot can appear quite normal even to careful examination until the child starts weight-bearing. Then a definite varus of the forefoot presents itself, usually with over-activity of the abductor hallucis muscle, so that the first toe in particular swings into medial position, with the inturn of the entire forefoot notably increased in comparison with the resting position.

Perhaps the lack of attention to this condition lies in the fact that the literature contains reports stating that normal alignment is reached by the end of the first year.[5] In attempting to explain the cause of foot strain in adults and to understand why one individual might suffer from painful feet with or without such extrinsic evidences as corns, calluses, bunions, clawing, inversions or eversions, muscular spasm, and increasing loss of flexibility, while others can wear any type of shoe without discomfort, we became more interested in deviations from normal in children's feet. By careful roentgen examination of adult feet, we can rule out such congenital abnormalities as accessory scaphoids, coalitions, or specific disease which may be found in a small percentage of patients. However, in most instances, when dealing with the chronically painful foot, we can find variations in the forefoot consistent with failure of full evolutionary development.

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The human foot is probably derived from a primitive mammalian foot, retaining its primitiveness supposedly because of the terrestrial habits. Jones[1] has stated the opinion that such a foot belongs to the primitive members of the primate stem, the stem from which man and the other existing members of the primates branched apart and went their separate ways at a very early stage. With the orthograde posture the feet were forced to take on the weight of the body.[2] This demanded an increase in the tarsus both in weight and in size, while the metatarsals and phalanges shortened. The opposing first toe came to lie beside the second. The changes, therefore, are (1) a progressive reduction of the anterior section (metatarsophalanges) and an increase in the posterior section (tarsus); (2) a change in the relative length of the digits, especially a reduction of the dominant third and an increase in the size of the first; (3) a gradual decrease in the abduction of the first and incorporation into the main structure of the foot; (4) a downward and backward extension of the os calcis to form a true heel and hence the appearance of the true arch; (5) a notable increase in the rigidity of the foot to fit it for its new purpose.

Therefore, in the normal foot which has developed completely according to evolutionary plan, Morton[4] has shown that the weight of the body is borne by the heel and the metatarsal heads. The static stresses are borne by the rigid framework of the foot, which is made up of bones and ligaments, while the muscles supply the kinetic power, and aside from keeping the body in balance, act only during locomotion. The weight of the body on the two feet can be divided into 24 units, each foot supporting 12 units. These are equally divided between the os calcis posteriorly and the metatarsal heads anteriorly. The wide first metatarsal head

rests on two sesamoid bones supporting one unit, thus making six points of contact with the ground. The foot then represents a triangle with its apex at the os calcis and the opposite side formed by the metatarsal heads. A line drawn from the os calcis should pass between the second and third metatarsal bones in erect standing, thus dividing equally the weight-bearing areas. In running, the full weight of the body will be thrust upon the metatarsal head of one foot (24 units), half of this being thrust upon the first metatarsal head alone. Then a line from the os calcis falls between the first and second metatarsals.

The weight of the body is transmitted through the astragalus (talus) to the os calcis, which forms a table upon which it rests. The bones of the midtarsal region represent a dome-shaped transverse arching, increasing the structural strength for forces applied vertically and converging the stresses toward the separate metatarsals, which are arched proximally but flattened distally so that their heads lie in a single plane.

The first metatarsal then is of particular importance to the internal mechanism of the foot: (1) It supports the medial side of the foot and prevents depression of the sustentaculum tali, thus preventing pronation. (2) It supports a major share of static stresses in locomotion. (3) It gives support to the astragalus, which is the center from which static stresses are distributed.

Any failure of normal evolutionary development, then, which interferes with the stability of the first metatarsal or changes the balance of weight-bearing so that the foot cannot conform to shoes or to long standing on hard surfaces is a potential cause for strain.

Barring severe trauma, paralyses, infections, etc., true foot strain can be attributed to certain deviations from the so-called normal foot which are inherited and represent a failure of full evolutionary development. Morton has defined certain anatomic variations which lead to definite strain: (1) a short first metatarsal, or (2) posteriorly placed sesamoids under the first metatarsal, and (3) hypermobility of the first metatarsal segments (including the first cuneiform bone which moves with the metatarsal). The short heel cord is considered as an extrinsic factor. He does not mention the metatarsus primus varus which is seen so frequently, but it does belong under the heading of "hypermobility of the first metatarsal segment." It has been discussed by many authors. Lapidus[3] described this condition as a primary cause of hallux valgus, since a large degree of angulation (10 to 19 degrees) exists between the first and second metatarsals with the first metatarsal in abduction.

In addition, I wish to add still another factor closely related to these as a cause of strain, namely, metatarsus varus, or a varus of the entire forefoot. This condition does persist into adulthood and can be easily demonstrated roentgenologically. Except by careful x-ray examination, it is difficult to recognize even when it is suspected, because the angle between the first and second metatarsals is normal since the entire forefoot is in varus and sufficient adjustment takes place in the tarsus to give a fairly straight medial line to the foot. We find, then, that in a large percentage of cases of foot strain in adults, the intrinsic causes are: (1) short first metatarsal; or posteriorly placed sesamoids under the first metatarsal; or hypermobility of the first metatarsal segment; (2) metatarsus varus, with or without metatarsus primus varus; and (3) the very important extrinsic factor—short heel cord. If individuals having feet of this type were not forced to wear shoes never designed for them, or to walk on hard surfaces which give no flexible support to the first metatarsal, then this deviation from the so-called normal would have little or no importance as these are fundamentally strong feet, failing only of full evolutionary development. The abnormalities are of course familial.

Recognizing that metatarsus varus does not disappear in childhood but persists in the adult foot, the question arises as to what correction should be attempted in infants. The amount of metatarsus varus in babies varies tremendously. Two infants in a series observed by the author gave the appearance of having clubbed feet, showing not only a pronounced varus deformity but the inversion of the tarsus and the short heel cords as well. The ease with which the last two elements were corrected with casts seemed enough to rule out true club-foot, but the varus deformity in one child has remained to a considerable degree; the feet of the other child show good correction at two years of age but the child is still wearing corrective shoes. The correction of the feet of the first child should have been much better had not an attempt been made to use leather molds after casting for only one month.

Pediatricians are realizing that routine development does not overcome the varus deformity but rather that it deserves special consideration. In spite of this, frequently babies with the deformity are not referred for orthopedic consultation until they begin to walk. Certain pediatricians who have been particularly alert to this condition, and who ordinarily refer patients for early care, have stated that the feet of those whom they referred late, appeared normal until weight-bearing began. Usually, the condition can be recognized immediately after delivery, and treatment should be started within the first two to three weeks. This simplifies the care as the child is ready for shoes by the time it is ready to walk, and it does not have to be constrained by casts with all the attending increase in care after walking is begun.

The author has placed casts on 32 of 60 babies observed for metatarsus varus and improvement has resulted. If it is decided that a correction should be forced, then casts are necessary, as no other simple support, such as a leather mold, will produce a shift in the forefoot. Casting is started at as early an age as possible and carried on for at least four or

five months. The casts are applied one week and wedged open on the medial side at the tarso-metatarsal junction the next week. They are changed completely every two weeks. Inasmuch as this condition is an inherited characteristic, the growth factor has to be considered, and, though the varus deformity can be corrected, if a wide angle is present between the first and second metatarsals this tends to persist.

When the condition is severe, there is often a tight medial band of soft tissue which is hard to stretch. When the varus is marked, and particularly when this medial band is present, casts should be used until appropriate shoes can be fitted. Although the first metatarsal may not entirely lose its broad angle with the second, the whole forefoot will be brought into better line with the tarsus and the ratio of weight-bearing improved. This in itself will permit the wearing of a more usual shoe. After the casts are removed, as the child is beginning to walk, shoes can be fitted to the opposite foot. Later the pronator type of shoe can be used or shoes of good quality can have a 1/8 to 3/8-inch lift on the lateral part of the sole and medial portion of the heel to thrust the forefoot into line with the tarsus. It is particularly important that the shoes be closely and accurately fitted. They should be worn at night as well as during the day.

When the varus deformity is of a moderate degree and passive correction is easily obtained, stretching by grasping the tarsus and moving the metatarsals lateralward at the tarsometatarsal junction should be done many times each day. When firm shoes can be fitted, they can be applied with the corrections described previously.

SUMMARY AND CONCLUSIONS

Metatarsus varus should receive more attention by pediatricians since it may not disappear but is frequently seen in adult feet and is a cause of strain and deformity. It can be present with metatarsus primus varus, with Morton's syndrome of short first metatarsal and hypermobility of the first metatarsal segment, or it can be present without evidence of these, in which case it is impossible to recognize without roentgenograms.

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